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APPLICATION NO.	FILING DATE	FIRST NAMED INVENTOR	ATTORNEY DOCKET NO.	CONFIRMATION NO.
09/904,181	07/11/2001	Michael W. Leviten	R-456	1164

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DELTAGEN, INC.
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EXAMINER

PARAS JR, PETER

ART UNIT

PAPER NUMBER

1632

DATE MAILED: 06/06/2002

9

Please find below and/or attached an Office communication concerning this application or proceeding.

Office Action Summary

Application No.

09/904,181

Applicant(s)

LEVITEN, MICHAEL W.

Examiner

Peter Paras

Art Unit

1632

-- The MAILING DATE of this communication appears on the cover sheet with the correspondence address --

Period for Reply

A SHORTENED STATUTORY PERIOD FOR REPLY IS SET TO EXPIRE 3 MONTH(S) FROM THE MAILING DATE OF THIS COMMUNICATION.

- Extensions of time may be available under the provisions of 37 CFR 1.136(a). In no event, however, may a reply be timely filed after SIX (6) MONTHS from the mailing date of this communication.
- If the period for reply specified above is less than thirty (30) days, a reply within the statutory minimum of thirty (30) days will be considered timely.
- If NO period for reply is specified above, the maximum statutory period will apply and will expire SIX (6) MONTHS from the mailing date of this communication.
- Failure to reply within the set or extended period for reply will, by statute, cause the application to become ABANDONED (35 U.S.C. § 133).
- Any reply received by the Office later than three months after the mailing date of this communication, even if timely filed, may reduce any earned patent term adjustment. See 37 CFR 1.704(b).

Status

- 1) ☐ Responsive to communication(s) filed on 12 April 2002.
- 2a) ☐ This action is **FINAL**. 2b) ☒ This action is non-final.
- 3) ☐ Since this application is in condition for allowance except for formal matters, prosecution as to the merits is closed in accordance with the practice under *Ex parte Quayle*, 1935 C.D. 11, 453 O.G. 213.

Disposition of Claims

- 4) ☐ Claim(s) 1-25 is/are pending in the application.
- 4a) Of the above claim(s) 1-4, 11, 13-16 and 21-25 is/are withdrawn from consideration.
- 5) ☐ Claim(s) _____ is/are allowed.
- 6) ☒ Claim(s) 5-10, 12 and 17-20 is/are rejected.
- 7) ☐ Claim(s) _____ is/are objected to.
- 8) ☐ Claim(s) _____ are subject to restriction and/or election requirement.

Application Papers

- 9) ☐ The specification is objected to by the Examiner.
- 10) ☐ The drawing(s) filed on _____ is/are: a) ☐ accepted or b) ☐ objected to by the Examiner.
Applicant may not request that any objection to the drawing(s) be held in abeyance. See 37 CFR 1.85(a).
- 11) ☐ The proposed drawing correction filed on _____ is: a) ☐ approved b) ☐ disapproved by the Examiner.
If approved, corrected drawings are required in reply to this Office action.
- 12) ☐ The oath or declaration is objected to by the Examiner.

Priority under 35 U.S.C. §§ 119 and 120

- 13) ☐ Acknowledgment is made of a claim for foreign priority under 35 U.S.C. § 119(a)-(d) or (f).
a) ☐ All b) ☐ Some * c) ☐ None of:
1. ☐ Certified copies of the priority documents have been received.
2. ☐ Certified copies of the priority documents have been received in Application No. _____.
3. ☐ Copies of the certified copies of the priority documents have been received in this National Stage application from the International Bureau (PCT Rule 17.2(a)).
* See the attached detailed Office action for a list of the certified copies not received.
- 14) ☒ Acknowledgment is made of a claim for domestic priority under 35 U.S.C. § 119(e) (to a provisional application).
a) ☐ The translation of the foreign language provisional application has been received.
- 15) ☐ Acknowledgment is made of a claim for domestic priority under 35 U.S.C. §§ 120 and/or 121.

Attachment(s)

- 1) ☒ Notice of References Cited (PTO-892)
- 2) ☐ Notice of Draftsperson's Patent Drawing Review (PTO-948)
- 3) ☒ Information Disclosure Statement(s) (PTO-1449) Paper No(s) 6
- 4) ☐ Interview Summary (PTO-413) Paper No(s) _____
- 5) ☐ Notice of Informal Patent Application (PTO-152)
- 6) ☐ Other: _____

DETAILED ACTION

Election/Restrictions

Applicant's election with traverse of Group II, claims 5-10, 12, and 17-20, in Paper No. 8 is acknowledged. The traversal is on the ground(s) that the Examiner has not shown that a serious burden would be required to examine all the claims. This is not found persuasive because each of the Inventions requires a separate search status. In particular, it is maintained that the products of Groups I, II, IV, and V are different each from the other; they each have different chemical structures and can be used in materially different methods that require different technical considerations. For example, the DNA targeting construct of Group I can be used to disrupt a ubiquitin-specific protease gene in a somatic cell *in vitro*, the transgenic non-human animal of Group II can be used as a model of disease, the unknown agents of Group IV can be used for modulating the expression of a ubiquitin-specific protease in a somatic cell *in vitro*, and the agent of Group V can be used for modulating the function of a ubiquitin-specific protease gene. It is maintained that the products of Inventions I, II, IV and V are distinct due to their divergent subject matter (DNA targeting construct, transgenic non-human animal, unknown agent that can modulate the expression of a ubiquitin-specific protease, an agent that can modulate the function of a ubiquitin-specific protease gene) and are separately classified and searched.

It is maintained that the methods of Groups III and VI are distinct, comprising different methodologies and using different products. For example, the method of Group III can be practiced in a somatic cell *in vitro*, while the method of Group VI is

required to be practiced in a transgenic non-human animal. It is maintained that the methods of Groups III and VI are distinct as they are directed to different methods that require the use of different products that need different technical considerations (somatic cells *in vitro* and transgenic non-human animals) and are separately searched and classified.

It is maintained that the products of Groups I, II, IV and V are distinct from the methods of Groups III and VI; the products of Groups I, II, IV, and V can be used in methods, which require different reagents and technical considerations from the methods of Groups III and VI. For example, the DNA targeting construct of Group I may be used as a probe in a hybridization assay *in vitro* while the transgenic non-human animal of Group II may be used to produce antibodies to an antigen, while the method of Group III may be used to identify agents that modulate the expression of a ubiquitin-specific protease. The method of Group III may be practiced with agents that have different chemical structures from the agents of Groups IV and V. It is maintained that the products of Groups I, II, IV and V are distinct from and can be used in different methods (hybridization assays, generating antibodies) from the methods of Groups III and VI.

Therefore it is maintained that all the inventions are distinct each from the other for the reasons given above. The requirement is still deemed proper and is therefore made FINAL.

Please note that after a final requirement for restriction, the Applicants, in addition to making any response due on the remainder of the action, may petition the

Commissioner to review the requirement. Petition may be deferred until after final action on or allowance of claims to the invention elected, but must be filed not later than appeal. A petition will not be considered if reconsideration of the requirement was not requested. (See § 1.181.).

Claims 1-4, 11, 13-16, and 21-25 are withdrawn from further consideration pursuant to 37 CFR 1.142(b), as being drawn to a nonelected Invention, there being no allowable generic or linking claim. Applicant timely traversed the restriction (election) requirement in Paper No. 8.

Priority

Applicant has not complied with one or more conditions for receiving the benefit of an earlier filing date under 35 U.S.C. 119(e) as follows:

An application in which the benefits of an earlier application are desired must contain a specific reference to the prior application(s) in the first sentence of the specification or in an application data sheet (37 CFR 1.78(a)(2) and (a)(5)).

The first sentence of the specification does not provide the serial of the provisional application filed on June 26, 2001. The attorney docket number provided is not sufficient to perfect priority.

Claim Rejections - 35 USC § 112, 1st paragraph

The following is a quotation of the first paragraph of 35 U.S.C. 112:

The specification shall contain a written description of the invention, and of the manner and process of making and using it, in such full, clear, concise, and exact terms as to enable any person skilled in the art to which it pertains, or with which it is most nearly connected, to make and use the same and shall set forth the best mode contemplated by the inventor of carrying out his invention.

Claims 5-10, 12 and 17-20 are rejected under 35 U.S.C. 112, first paragraph, because the specification, while being enabling for a transgenic mouse comprising a disruption of the nucleotide sequence set forth in SEQ ID NO: 1, wherein the mouse exhibits a phenotype of increased PPI, cells obtained from the same transgenic mouse, and methods of screening agents using the same transgenic mouse does not reasonably provide enablement for all other transgenic non-human animals, cells obtained from the same, methods of using the same and any cell comprising a disruption in a ubiquitin-specific protease gene. The specification does not enable any person skilled in the art to which it pertains, or with which it is most nearly connected, to make and use the invention commensurate in scope with these claims.

The claims are directed transgenic non-human animals, particularly a mouse, that comprise a disruption in a ubiquitin-specific protease gene. The claims are further directed cells obtained from the same transgenic non-human animals, a method of producing the same transgenic mouse, and a method of screening agents using the same. The claims are also directed to cells, particularly murine embryonic stem cells that comprise a disruption in a ubiquitin-specific protease gene

The specification teaches the generation of transgenic mice by disruption of the nucleotide sequence set forth in SEQ ID NO: 1. See pages 6, lines 3-14, and the working example on page 48, lines 6-24 of the specification. The specification teaches that these knockout mice, even as heterozygotes, exhibit a specific phenotype, of increased prepulse inhibition (PPI) with a 90dB prepulse, as a result of the disruption of the nucleotide sequence set forth in SEQ ID NO: 1. See page 49 of the specification.

While the specification has taught the generation of such a transgenic knockout mouse having a phenotype of increased PPI, the specification has not taught the generation of the other transgenic non-human animals comprising a disruption in a ubiquitin-specific protease gene encompassed by the claims. The working examples, guidance and relevant teachings provided by the instant specification are directed to the creation of the above transgenic mouse but do not support the creation of other transgenic non-human animals encompassed by the claims. See pages 48-49.

With regard to claim breadth, the standard under 112, first paragraph entails the determination of what the claims recite and what the claims mean as a whole. In addition, when analyzing the enabled scope of the claims, the teachings of the specification are taken into account because the claims are to be given their broadest reasonable interpretation that is consistent with the specification. As such, in light of the specification, the claimed invention is properly interpreted with regard to the disclosed phenotype of the exemplified transgenic mice comprising a disruption of the nucleotide sequence set forth in SEQ ID NO: 1. Such an interpretation is consistent with the specification despite that the claimed non-human mammals require only that they comprise a disrupted ubiquitin-specific protease gene. This is because, with regard to the enablement requirement, one of skill in the art must be provided with both how to make and use the claimed invention. As such, the enabled scope of the claimed invention, in light of the teachings of the specification, is found to be the generation of transgenic mice comprising a disruption of the nucleotide sequence set forth in SEQ ID NO: 1 which exhibit a phenotype of increased PPI.

The following aspect of the rejection under 35 U.S.C. 112, first paragraph is directed to claims 5-9 and 12 as they read on embryonic stem cells and transgenic knockout non-human animals:

For the purpose of this rejection claims 5-6 are interpreted to encompass embryonic stem cells. The specification has not provided any other uses for embryonic stem cells than for the creation of transgenic non-human animals.

Both the specification and the state of the art have taught that the transgenic knockout technology requires the use of embryonic stem cells that have been genetically manipulated to comprise a disruption in a nucleotide sequence of interest. The specification has not taught creation of a transgenic knockout non-human animal by methods that do not require embryonic stem cells. Presently, the transgenic knockout technology is limited to the mouse system. See below.

With regard to the claim breadth directed to transgenic non-human animals, the specification fails to teach the production of any transgenic non-human animal comprising a disruption in a ubiquitin-specific protease gene other than a transgenic knockout comprising a disruption in the nucleotide sequence set forth in SEQ ID NO: 1. It is well known in the knockout art that the production of knockout animals other than mice is undeveloped. This is because ES cell technology is generally limited to the mouse system, at present, and that only "putative" ES cells exist for other species. See Moreadith et al. at page 214, Summary. Seamark (Reproductive Fertility and Development, 1994) supports this observation by reporting that totipotency for ES cell technology in many livestock species has not been demonstrated (page 6, Abstract).

Likewise, Mullins et al. (Journal of Clinical Investigation, 1996) state that "although to date chimeric animals have been generated from several species including the pig, in no species other than the mouse has germline transmission of an ES cell been successfully demonstrated." (page S38, column 1, first paragraph). As the claims are directed to transgenic non-human animals and cells derived therefrom (claims 8-9) or a method that requires the use of a transgenic non-human animal (claim 12) or interpreted to read on embryonic stem cells (as in claims 5-6) comprising a disruption in a ubiquitin-specific protease gene, which must be generated by the introduction of a transgene into an ES cell, the state of the art supports that only mouse ES cells were available for use for production of transgenic mice. Finally, claim 7 is appropriately rejected in this section as the term murine, as defined in a dictionary, encompasses both rats and mice. The state of the art does not support the use of rat embryonic stem cells for creating knockout rats. See above. Given the unpredictable state of the art it would have required undue experimentation for the skilled artisan to create transgenic knockout non-human animals of species other than the mouse.

Claims 8, 12, and 20 encompass transgenic non-human animals that comprise a disruption in a ubiquitin-specific protease gene that do not exhibit any particular phenotype. The state of the art at the time of filing was such that one of skill could not predict the phenotype of a knockout mouse (Moreadith et al., 1997, J. Mol. Med., Vol. 75, pages 208-216; see page 208, column 2, last full paragraph). Moens et al. (Development, Vol. 119, pages 485-499, 1993) disclose that two mutations produced by homologous recombination in two different locations of the N-myc gene produce two

different phenotypes in mouse embryonic stem cells, one leaky and one null (see abstract). The specification has asserted that the nucleotide sequence set forth in SEQ ID NO: 1 encodes a ubiquitin-specific protease. However, as the ubiquitin-specific protease family comprises many members and the specification has not provided any guidance as to which type of ubiquitin-specific protease is encoded by the nucleotide sequence set forth in SEQ ID NO: 1 it would be difficult to predict any phenotype resulting from disruption of the sequence of SEQ ID NO: 1. The specification discloses a phenotype exhibited by knockout mice comprising a disruption in the nucleotide sequence set forth in SEQ ID NO: 1 is increased prepulse inhibition with a 90dB prepulse. See page 49 of the specification. Claims 8, 12, and 20, as written, do not include a phenotype that differs from the wild-type mouse. Moreover the skilled artisan would know how to use a transgenic knockout non-human animal that lacks a phenotype, particularly because the instant specification has not provided uses for such; the transgenic mice that have a phenotype of increased PPI may be used for drug testing according to the instant specification. The specification overcomes the unpredictability in obtaining a phenotype associated with a disruption of the nucleotide sequence set forth in SEQ ID NO: 1; however, the claims are not commensurate in scope with the enabled phenotype disclosed in the specification. Inclusion of a phenotype associated with a disruption of the nucleotide sequence set forth in SEQ ID NO: 1 in a mouse in the claims would overcome this aspect of the rejection. Given the unpredictable nature of a phenotype that results from disruption of a nucleotide

sequence it would have required undue experimentation for the skilled artisan to use a transgenic non-human knockout animal that lacks a phenotype.

With regard to claim 12, it appears that the claim as written is not enabled. The claim is directed to a method of identifying an agent that modulates the function of a ubiquitin-specific protease. The steps of the method however do not enable the claim because a disrupted ubiquitin-specific protease gene may not produce any functional protein. As such it is not possible to modulate a non-existent protein. Further, the claim as written does not correlate modulation of a disrupted ubiquitin-specific protease gene with modulation of a ubiquitin-specific protease. It is unclear and unpredictable how modulation of a disrupted gene correlates to modulation of a protein. The evidence of record does not provide support for such a correlation.

As a final issue, claims 5-10, 12, and 17-20 encompass non-human animals and cells comprising a disruption in a ubiquitin-specific protease gene. The specification has disclosed mice and cells that comprise a disruption in the nucleotide sequence set forth in SEQ ID NO: 1. The specification has asserted that the nucleotide sequence set forth in SEQ ID NO: 1 encodes a ubiquitin-specific protease. The state of the art however suggests that the family of ubiquitin-specific proteases comprises many members that have different structures and functions (i.e. they act on different substrates). See Finley et al [1991, Annual Review of Cell Biology, Ubiquitination, pages 25-69, provided in Applicant's IDS] who discuss the structures and functions of various enzymes of the ubiquitin system across species. Also see the instant specification (on page 1, in lines 12-19), which reports on the diversity of the ubiquitin-

specific protease family. As such the claims broadly encompass disruption of nucleotide sequences encoding a whole family of proteases, the ubiquitin-specific proteases, which have different structures and functions. The instant specification, however, has not provided any evidence that the nucleotide sequence set forth in SEQ ID NO: 1 actually encodes a ubiquitin-specific protease. Although the specification has suggested that the nucleotide sequence set forth in SEQ ID NO: 1 shares sequence homology to other nucleotide sequences that encode known ubiquitin-specific proteases (see page 2, lines 5-6), the specification has failed to teach which sequences that encode ubiquitin-specific protease share homology with the nucleotide sequence set forth in SEQ ID NO: 1. As such, it is unclear which type of ubiquitin-specific protease is encoded by the nucleotide sequence of SEQ ID NO: 1.

Furthermore, no evidence has been presented which suggests that the protein product encoded by the nucleotide sequence set forth in SEQ ID NO: 1 functions as a ubiquitin-specific protease or which substrates it may act on. Limiting the claims to disruption of the nucleotide sequence set forth in SEQ ID NO: 1 may be sufficient to overcome this aspect of the rejection.

Therefore, in view of the quantity of experimentation necessary to determine the parameters listed above for the production of transgenic non-human animals comprising a disruption in a ubiquitin-specific protease gene, the lack of direction or guidance provided by the specification for the production of transgenic non-human animals comprising a disruption in a ubiquitin-specific protease gene, the absence of working examples for the demonstration or correlation to the production of a transgenic knockout

non-human animal that exhibits a phenotype other than the exemplified mouse, the unpredictable state of the art with respect to a phenotype that results from disruption of a given nucleotide sequence, the undeveloped art pertaining to the establishment of true embryonic stem (ES) cells of animal species other than mouse, and the breadth of the claim drawn to all non-human animals and all ubiquitin-specific protease, it would have required undue experimentation for one skilled in the art to make and/or use the claimed invention.

Claim Rejections - 35 USC § 112, 2nd paragraph

The following is a quotation of the second paragraph of 35 U.S.C. 112:

The specification shall conclude with one or more claims particularly pointing out and distinctly claiming the subject matter which the applicant regards as his invention.

Claims 5-7, and 12 rejected under 35 U.S.C. 112, second paragraph, as being indefinite for failing to particularly point out and distinctly claim the subject matter which applicant regards as the invention.

Claim 5 is indefinite as written. The claim is directed to a cell comprising a disruption in a ubiquitin-specific protease gene. However, the claim does not specify if the cell is *in vitro* or *in vivo*. The claim is indefinite because if the cell is interpreted to read on a cell *in vivo* it is not clear if Applicants intend to claim a cell within an animal or an animal. Amending the claim to read on an isolated cell may be sufficient to overcome this rejection. Claims 6-7 depend from claim 5.

Claim 12 is indefinite as written. The claim is directed to a method of identifying an agent that modulates the function of a ubiquitin-specific protease. The claim is indefinite because the method steps do not set forth the goal of the preamble for the following reasons: 1) step (a) requires a non-human transgenic animal comprising a disruption in a ubiquitin-specific protease gene however as a disruption of a ubiquitin-specific protease gene would prevent production of a ubiquitin-specific protease it is unclear how such can be modulated; and 2 step (c) requires determining whether the function of the disrupted ubiquitin-specific protease gene in the transgenic non-human animal is modulated however as the preamble of the claim is directed to modulation of a protein it is unclear how modulation of the function of a disrupted gene can set forth such a goal.

Claim Rejections - 35 USC § 103

The following is a quotation of 35 U.S.C. 103(a) which forms the basis for all obviousness rejections set forth in this Office action:

(a) A patent may not be obtained though the invention is not identically disclosed or described as set forth in section 102 of this title, if the differences between the subject matter sought to be patented and the prior art are such that the subject matter as a whole would have been obvious at the time the invention was made to a person having ordinary skill in the art to which said subject matter pertains. Patentability shall not be negated by the manner in which the invention was made.

Claims 5-10 are rejected under 35 U.S.C. 103(a) as being unpatentable over Capecchi (1994, Scientific American, pages 52-59) taken with Wood et al (Mechanisms of Development, 1997, 63: 29-38).

The claims are directed transgenic non-human animals that comprise a disruption in a ubiquitin-specific protease gene. The claims are further directed cells obtained from the same transgenic non-human animals, and a method of producing the same transgenic non-human animal. The claims are also directed to cells, particularly murine embryonic stem cells that comprise a disruption in a ubiquitin-specific protease gene

Capecchi teaches knockout technology applied to mice specifically with respect to the disruption of the *HoxA-3* gene and as the method of producing the same applies to determining the *in vivo* biological function of any known gene of interest. See page 59. For example, Capecchi discloses the applicability of gene targeting to many other genes so that a correlation can be drawn between the malfunctioning of the gene to the manifestation of disease (See page 54, columns 1-3). Capecchi further discloses the essential components of a targeting vector [(page 54, fig. 1 and page 57, column 1, paragraph 2), such as nucleotide sequences, which are homologous to endogenous genomic nucleotide sequences, that flank a selection marker, such as the neomycin resistance gene] and the steps involved for targeted gene replacement in ES cells as well as in mice (pages 55-56 and diagrams). Capecchi differs from the claimed invention by not teaching transgenic non-human animals and cells that comprise a disrupted ubiquitin-specific protease gene.

However at the time the claimed invention was made, Wood et al teach the cloning of a mouse nucleotide sequence that encodes a putative ubiquitin-specific protease, Fam. See page 30, column 1, last paragraph and also the Experimental

procedures beginning on page 36, in column 2. Wood et al teach that FAM shares extensive sequence homology with a Drosophila ubiquitin-specific protease, faf. See Figure 2 on page 31, also section 2.1.1 beginning on page 31 and bridging to page 32, and also section 3.1 beginning on page 33 and bridging to page 35. In view of the extensive sequence similarity between Drosophila and mouse proteins Wood et al have suggested that Fam is the mouse ortholog of faf, and thus is a ubiquitin-specific protease also. See page 35, column 1 beginning with the first full sentence. Wood et al have suggested a role for Fam in the developing embryo based on mRNA expression analyses during development. See sections 2.3.1-2.3.5 on pages 32-33 and also figures 5-7 on page 34. Wood et al discuss that to date there has been very little investigation of the role played by the ubiquitin pathway in the development of multicellular organisms and that the discovery of Fam should help speed the elucidation of the role of the ubiquitin system in the development of multicellular organisms. See page 36, column 1, the last paragraph bridging to column 2. Note that absent any phenotypic requirements of the claimed transgenic non-human mammal, the combination of the cited prior art is sufficient to make obvious the claimed invention.

Accordingly, in view of the teachings of Wood et al., it would have been obvious for one of ordinary skill in the art, at the time the claimed invention was made, to modify the knockout technology of Capecchi by use of a targeting vector for disruption of the Fam gene in a mouse with a reasonable expectation of success. One of ordinary skill in the art would have been sufficiently motivated to make such a modification as it was an art-recognized goal to determine the physiological role of a gene of interest by the

generation of a knockout mouse as taught by Capecchi et al, and particularly since Wood et al suggest a need to investigate the role of the ubiquitin pathway in the development of multicellular organisms (see page 36, column 1, the first sentence of the last paragraph), and more particularly because Wood et al suggest that the discovery of Fam should help speed the elucidation of the role of the ubiquitin system in the development of multicellular organisms (see page 36, column 2, the last sentence of the first paragraph prior to the Experimental procedures).

Thus, the claimed invention, as a whole, is clearly *prima facie* obvious in the absence of evidence to the contrary.

Allowable Subject Matter

The following subject matter appears to be allowable over the prior art of record: a transgenic mouse whose genome comprises a disruption of the nucleotide sequence set forth in SEQ ID NO: 1 and has a phenotype of increased prepulse inhibition (PPI). Methods of using the same mouse for screening agents that may affect the phenotype and cells obtained from the same mouse also appear to be allowable over the prior art of record.

Conclusion

No claim is allowed. Claims 12, 17-20 appear to be free of the prior art of record but are subject to other rejections. See above.

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Any inquiry concerning this communication or earlier communications from the examiner(s) should be directed to Peter Paras, Jr., whose telephone number is 703-308-8340. The examiner can normally be reached Monday-Friday from 8:30 to 4:30 (Eastern time).

If attempts to reach the examiner by telephone are unsuccessful, the examiner's supervisor, Deborah Reynolds, can be reached at 703-305-4051. Papers related to this application may be submitted by facsimile transmission. Papers should be faxed via the PTO Fax Center located in Crystal Mall 1. The faxing of such papers must conform with the notice published in the Official Gazette, 1096 OG 30 (November 15, 1989). The CM1 Fax Center numbers are (703) 308-4242 and (703) 305-3014.

Inquiries of a general nature or relating to the status of the application should be directed to Patsy Zimmerman whose telephone number is (703) 305-2758.

Peter Paras, Jr.

Art Unit 1632

Peter Paras Jr
Art Unit 1632